Tracking Pandemic Severity Using Data on the Age Structure of Mortality: Lessons From the 1918 Influenza Pandemic in Michigan

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Objectives. To test whether distortions in the age structure of mortality during the 1918 influenza pandemic in Michigan tracked the severity of the pandemic.

Methods. We calculated monthly excess deaths during the period of 1918 to 1920 by using monthly data on all-cause deaths for the period of 1912 to 1920 in Michigan. Next, we measured distortions in the age distribution of deaths by using the Kuiper goodness-of-fit test statistic comparing the monthly distribution of deaths by age in 1918 to 1920 with the baseline distribution for the corresponding month for 1912 to 1917.

Results. Monthly distortions in the age distribution of deaths were correlated with excess deaths for the period of 1918 to 1920 in Michigan ($r = 0.83$; $P < .001$).

Conclusions. Distortions in the age distribution of deaths tracked variations in the severity of the 1918 influenza pandemic.

Public Health Implications. It may be possible to track the severity of pandemic activity with age-at-death data by identifying distortions in the age distribution of deaths. Public health authorities should explore the application of this approach to tracking the COVID-19 pandemic in the absence of complete data coverage or accurate cause-of-death data. (*Am J Public Health*. 2021;111(S2):S149–S155. https://doi.org/10.2105/AJPH.2021.306303)

As of February 22, 2021, the COVID-19 pandemic had claimed more than 500 000 lives in the United States.[1] According to Centers for Disease Control and Prevention mortality data,[2] it is likely that the 1-year total number of deaths attributable to COVID-19 will approach or even exceed 20% of the total number of deaths in the United States in 2020. While a precise count of the lives lost because of COVID-19 is impossible to compute, obtaining accurate estimates of the death toll across the globe is a matter of great importance for public health. Such estimates will enable us to better understand the epidemiology of COVID-19 and associations between various public health measures and pandemic outcomes.

Unfortunately, high-quality data on COVID-19 cases and COVID-19–related deaths are scarce. In most countries, the infrastructure for testing and diagnosing the disease and for accurately recording deaths by cause are inadequate. These gaps underscore the need for alternate and indirect methods to ascertain the severity of the pandemic across the world.

The influenza pandemic of 1918 to 1920 has become the benchmark against which the COVID-19 pandemic is compared. This influenza pandemic was the single most devastating pandemic in recent history, causing at least 50 million deaths,[3] including approximately 675 000 in the United States.[4] An effect of high mortality from the 1918 influenza and COVID-19 pandemics is their distortionary impact on demographic aggregates, including births[5,6] and deaths. In the case of COVID-19, mortality has been especially severe among elderly people, with case-fatality rates increasing with age.[7–9] In the case of the 1918 influenza, mortality was disproportionately high among young adults aged approximately 20 to 40 years (Figure 1).[10–12]

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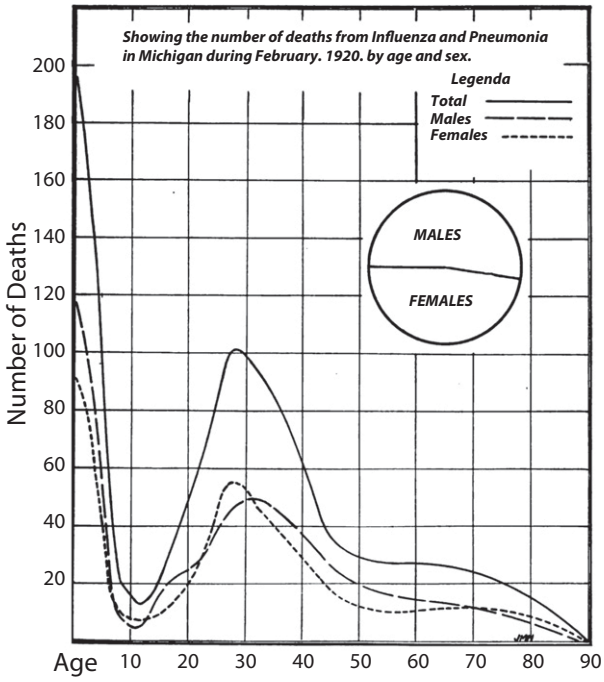

FIGURE 1— Number of Deaths From Influenza and Pneumonia in Michigan During February 1920 by Age

Source. Michigan Department of Health.[20] Digitally enhanced by Camille North.

Our aim, therefore, was to explore the degree to which distortions in the age structure of mortality that bear the signature of a pandemic disease can serve as an indicator of pandemic severity. To do so, we analyzed the age structure of mortality during the successive waves of excess mortality in Michigan over the 36 months comprising 1918 to 1920 and answered 3 questions: (1) Did age–mortality distributions for any of the 36 months show distortions consistent with the 1918 influenza? (2) Did the months identified in question 1 correspond with waves of excess mortality? (3) Is there an association between the severity of waves of excess mortality and the degree to which the age–mortality curve is distorted in comparison with the baseline age–mortality pattern? Our analysis demonstrated that the answer to each of these 3 questions is affirmative.

BACKGROUND

The age–mortality pattern of the 1918 influenza pandemic was "unique"[13] in that peak mortality was experienced by young adults in the 20- to 40-year age range,[14–16] leading researchers to characterize the pandemic as having a W-shaped age–mortality curve in contrast to the U-shaped curves usually seen in seasonal influenza.[11,17–19] Various theories have been advanced to explain this unusual pattern, including antigenic history (protective of older people), comorbidity with tuberculosis, heightened immune response (among younger adults), and T-cell dysregulation because of previous infection by another pathogen.[19] Michigan followed the W-shaped age–mortality pattern (Figure 1, for example).[20]

Some studies have found variations in infant and elderly mortality that do not fit the generalized W-shaped curve; this is likely attributable to small sample size (among the elderly)[21] and high baseline mortality (among children younger than 5 years) from noninfluenza causes.[22] Young adults, however, consistently experienced disproportionately high mortality. This "distinguishing feature"[11] creates a signature age–mortality curve (despite variations in infant and elderly mortality), which can be used as a proxy to identify possible pandemic-associated waves in the continuing absence of direct viral evidence.[11]

Studies variously identify the ages or age ranges of peak excess mortality or significant excess mortality during the 1918 influenza pandemic as 15 to 25,[13] 15 to 44,[23–25] 18 to 42,[17] 20 to 29,[22] 25 to 44,[14,18] and 28 years.[19] New York City, like Michigan, experienced multiple pandemic waves from spring 1918 to April 1920, with notably elevated mortality for ages 5 to 39 years[14] during all waves. Several previous studies have also identified elevated mortality risk among young adults during the spring 1918 wave,[13,14,23] suggesting that this was a herald wave caused by the same pathogen as the deadly fall 1918 wave. Studies on the winter 1919 wave and any 1920 waves have mixed age–mortality profiles.

METHODS

Our approach involved, first, computing monthly excess deaths in Michigan. Next, we analyzed the monthly age distributions of mortality for each of 36 months during the pandemic years (1918–1920) in relation to the baseline distribution corresponding to the month in question for 6 years preceding the pandemic (1912–1917) to look for shifts in the age composition of mortality consistent with the signature W-shaped pattern of the pandemic. We limited the pandemic

years to 1918 to 1920 because the consensus in previous studies is that the pandemic did not extend beyond 1920.[26] Third, we compared the timing of spikes in excess mortality with the timing of spikes in distortion of the age structure of mortality to ascertain whether they coincided. Finally, we used time-series methods to study monthly mortality by age group to identify the sources of distortion of the age structure of mortality in 1918 to 1920 during times of high distortion.

The data for this study contained monthly age distributions of deaths in Michigan between 1912 and 1920, for a total of 9 years (108 months). The variety of age ranges described in the previous section represents regional differences in reporting,[22] complicating both detailed age-distribution analysis and cross-study comparison. Reflecting the emphasis on vulnerable age categories, the Michigan Department of Health reported deaths in 4 age categories: younger than 1 year, 1 to younger than 5 years, 5 to younger than 65 years, and 65 years and older. This categorization allowed us to establish levels of young adult mortality that distinguished the 1918 pandemic, thereby enabling us to identify the likely influenza-associated excess mortality waves in Michigan coinciding with the pandemic.

The total number of deaths recorded for the entire period was 392 497.[27] Notably, the 3 vulnerable age groups (i.e., <1 year, 1 to <5 years, and ≥ 65 years) accounted for approximately two thirds of all total deaths (66%) even though they accounted for a disproportionately small portion of an average lifespan. For the pandemic years, the corresponding percentage had fallen to only 51%.

We computed monthly excess deaths in Michigan by seasonally adjusting the raw data on monthly deaths using the PROC X12 algorithm in SAS.[28] This algorithm adjusts a time series for regular cyclical fluctuations corresponding to a 12-month (seasonal) cycle using an iterative algorithm.[28,29] We used the additive variant of the algorithm. Given the presence of outliers during peak months of the pandemic, we also used the outlier detection feature, which eliminates distortions introduced into the algorithm when outliers are included in the computations.[28]

To compare the monthly age distributions of mortality in 1918 to 1920 with the prepandemic baseline, we aggregated the numbers of deaths in each age category by month for the years 1912 to 1917 to obtain the baseline age distribution of mortality. Next, we conducted 2 tests comparing the age distribution of mortality for each of the 36 months between January 1918 and December 1920 with the baseline age distribution for the corresponding month. One test, designed for ordered data, was the Kuiper test,[30] which modifies the better-known Kolmogorov–Smirnov test[31] by according greater weight to frequencies at the ends of the distribution (i.e., infants and the elderly). Given the nature of the data, in which the age categories at the ends of the distribution are of particular importance, we selected this test in preference to the standard Kolmogorov–Smirnov test. Briefly, the Kuiper statistic, K, is defined as[30]

$$K = D_+ + D_- \quad (1)$$

where D_+ is the supremum of the difference between the cumulative distribution functions of the 2 distributions and D_- is the infimum of the difference between the 2 cumulative distribution functions. We chose not to use the asymptotic Kuiper 2-sample test statistic because the scale factor for that statistic would be distorted for months with extremely high excess mortality, yielding an artificially high correlation.[32] We also compared the distributions using the standard χ^2 goodness-of-fit test.

Finally, we examined the evolution of excess deaths within each age category over time. Using the PROC X12 algorithm in SAS,[28,29] we adjusted the time series for regular seasonal patterns, thereby extracting the trend and irregular components of the age-specific counts of deaths. This approach complements the previous cross-sectional analysis with a time-series dimension.

RESULTS

Distortions in the age structure of mortality closely tracked excess mortality coinciding with the severity of the 1918 influenza pandemic. Figure 2a shows estimates of monthly excess mortality in 1918 to 1920. Figure 2b shows the χ^2 and Kuiper test statistics comparing the monthly age distributions of mortality for 1918 to 1920 with the baseline age distribution of mortality for that month. The similarity between the 2 graphs is striking. The peaks of excess mortality in Michigan (Figure 2a) coincide neatly with peaks in the Kuiper statistic and the χ^2 statistic (Figure 2b). The Kuiper test of the null hypothesis of no difference between the distribution for pandemic months and the corresponding baseline months is rejected for the following 9 months of high excess mortality: April, October, November, and December 1918; January and February 1919; and January, February, and March 1920. Notably, the null hypothesis was rejected for only 1 other month during this 36-month time period, July 1918. The correlation coefficient between the Kuiper statistic and excess mortality for the 36 months from January 1918 to December 1920 was 0.83 ($P < .001$). Even after the removal of 4

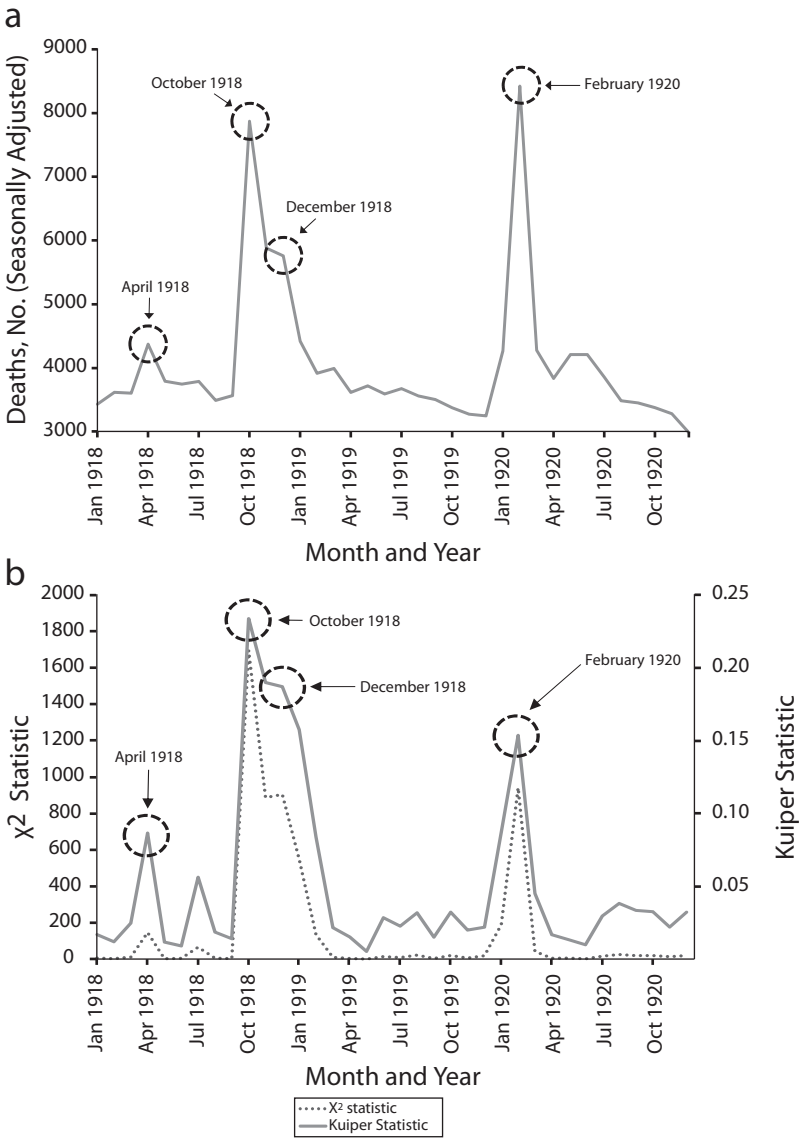

FIGURE 2— Monthly (a) Number of Deaths and (b) Test Statistics Comparing Age Distribution of Mortality vs Baseline: Michigan 1918–1920

Note. Data were seasonally adjusted.

outliers for excess mortality (October–-December 1918, February 1920), which is in any case not warranted because of their substantive importance, the coefficient was 0.52 ($P = .003$).

Table 1 demonstrates that distortions in the age structure of mortality for all 9 months of high excess mortality were similar. Without exception, deaths among infants and elderly people were proportionately lower during these peaks compared with normal years, and deaths among younger adults were proportionately higher.

Finally, Figure 3, showing seasonally adjusted deaths over time for each of the 4 age groups, reveals an interesting phenomenon. While the first peak of excess mortality (spring 1918) was accompanied by a noticeable spike in deaths among younger adults, the second peak of 1918 (fall and winter) showed spikes in deaths for younger adults and children, and the February 1920 peak resulted from spikes in excess mortality for all 4 age groups.

DISCUSSION

The results motivate 3 key observations. First, the timing of waves and peaks of excess mortality closely track distortions in the age structure of mortality. The high correlation between the degree to which the age–mortality distribution deviates from the monthly baseline and our estimates of excess pandemic-associated mortality suggests that these distortions, measured by the Kuiper statistic, may, under the right conditions, be a good measure of pandemic activity. These conditions include (1) accurate and representative, though not necessarily complete, data on the age structure of mortality; (2) timing that coincides with a known pandemic; and (3) the absence of other events that may distort the age structure of mortality.

Second, distortions in the age structure of mortality in Michigan uniformly resulted from a higher proportion of younger adults dying. This observation aligns with the widely observed W-shaped age structure of mortality in Michigan and other locations during the pandemic.

And third, with each successive peak of excess mortality, increasing numbers of people outside the worst-affected age group (ages 5 to < 65 years) were being affected. Figure 3 demonstrates that successive peaks of excess mortality expanded across age categories until, during the early 1920 wave, a spike in excess mortality was noticeable in each of the 4 age categories. This phenomenon raises the question of whether the 1920 wave was caused by the same or a different pathogen or, possibly, by separate pandemic and seasonal strains

TABLE 1— Age Distribution of Deaths During Months of High Excess Mortality: Michigan, 1918–1920

Time Period[a]	Percentage of Total Deaths by Age Category			
	0 to < 1 Year	1 to < 5 Years	5 to < 65 Years	≥ 65 Years
April 1918	14.35	5.24	52.54	27.87
April baseline	17.27	6.07	43.88	32.78
Variation from baseline	−2.92	−0.83	8.66	−4.91
October 1918	10.64	7.75	66.31	15.30
October baseline	18.06	5.98	44.70	31.26
Variation from baseline	−7.42	1.78	21.61	−15.96
November 1918	11.39	9.23	61.44	17.94
November baseline	15.88	5.27	46.40	32.44
Variation from baseline	−4.50	3.96	15.04	−14.50
December 1918	11.02	7.36	61.96	19.66
December baseline	16.03	5.08	45.53	33.36
Variation from baseline	−5.01	2.28	16.43	−13.70
January 1919	13.48	7.01	56.87	22.64
January baseline	16.34	5.06	43.06	35.55
Variation from baseline	−2.85	1.95	13.81	−12.91
February 1919	15.98	7.14	49.75	27.13
February baseline	17.15	5.25	43.24	34.36
Variation from baseline	−1.16	1.88	6.51	−7.23
January 1920	15.15	7.42	49.42	28.02
January baseline	16.34	5.06	43.06	35.55
Variation from baseline	−1.19	2.36	6.36	−7.53
February 1920	13.88	8.85	55.01	22.26
February baseline	17.15	5.25	43.24	34.36
Variation from baseline	−3.27	3.60	11.77	−12.10
March 1920	16.16	6.83	46.40	30.61
March baseline	17.52	5.70	43.01	33.77
Variation from baseline	−1.36	1.13	3.39	−3.16

[a]Baseline monthly data computed for the period 1912 to 1917.

simultaneously circulating among the population.[17] Alternatively, if, indeed, each wave was caused by the same H1N1 virus, why were varying cross-sections of the population being affected by each successive wave?[22] Was the virus evolving, was the surviving population developing immunity,[19,22,25,33] or was some behavioral factor such as the return of American troops to the United States in the aftermath of World War I responsible?

Limitations

The results of this study should be interpreted keeping in mind several limitations. First, the original data on deaths are limited to 4 age groups, ruling out a fine-grained analysis of age-related impacts. However, this level of aggregation enables us to focus on traditionally vulnerable age segments of the population. Furthermore, given the variation of excess mortality across age groups within the young adult category, as seen in earlier studies,[13,14,17,18,22,23] the data also demonstrate that aggregation does not appear to weaken the association between excess mortality and distortion of the age structure of mortality. A second limitation is the use of mortality rather than morbidity data to capture pandemic severity. This choice was made in line with the large literature on the 1918 pandemic that has used mortality because of the superior quality of such data. A third limitation is the excess mortality computation to attribute deaths to the pandemic, a necessary step because of the absence of accurate diagnostic testing at the time. This study follows previous work on the 1918 influenza pandemic using excess mortality to infer pandemic-associated excess deaths.[34] A fourth limitation of this study is the open question of whether the 1920 wave of influenza-like illness was caused by the same novel influenza virus as the 1918 to 1919 waves.[35]

Conclusions

In this study, we combined the findings of the cross-sectional analyses of age distributions of mortality with the time-series analyses of mortality by age group to characterize the age structure of mortality during the successive waves of excess mortality coinciding with the timing of the 1918 influenza pandemic in Michigan. We found a striking pattern of similarity between the degree to which the age structure of mortality for high-excess-mortality months between 1918 and 1920 deviated from the monthly norm and the magnitude of excess mortality during those months. The highly synchronous pattern of these distortions with excess mortality (Figures 2a and 2b) suggests that

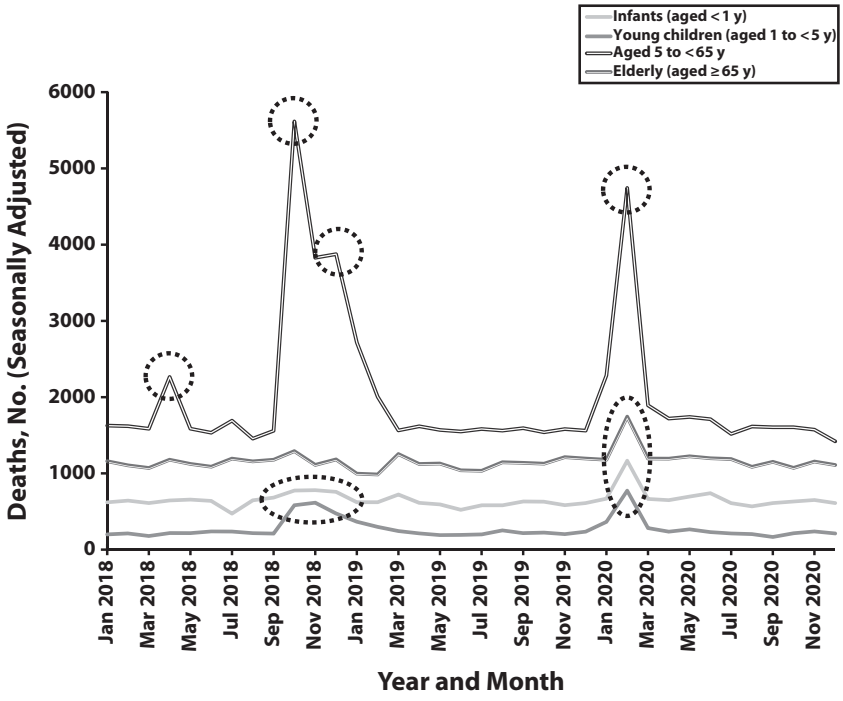

FIGURE 3— Monthly Deaths by Age Group: Michigan, 1918–1920

information about the age structure of mortality may convey valuable information about pandemic activity.

An important implication of this study is that, even with incomplete data coverage or in the absence of detailed cause-of-death data during pandemics such as the ongoing COVID-19 pandemic, it may be possible for researchers to detect and estimate pandemic activity by using age-at-death data to identify distortions of the age structure of mortality consistent with elevated pandemic-associated mortality. Given the inadequacy of data collection, reporting, and diagnostic systems in many areas, an indirect method such as this may enable us to better characterize the epidemiology of pandemics, thereby strengthening our understanding of them and ways in which to mitigate their devastating consequences. With this in mind, we hope that future research will closely examine the age structure of mortality of the COVID-19 pandemic and explore the use of distortions in this age structure to identify locations around the world where the pandemic may have struck unnoticed, unrecorded, or underestimated. AJPH

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CONTRIBUTORS

S. Chandra conceptualized and designed the project, collected and analyzed the data, interpreted the results, and co-wrote all sections of the article. J. Christensen assisted in conceptualization and design of the project, collection and analysis of the data, and interpretation of the results, and co-wrote all sections of the article.

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CONFLICTS OF INTEREST

Neither author has conflicts of interest to report.

HUMAN PARTICIPANT PROTECTION

The research reported in this article did not involve human participants. Therefore, institutional review board approval was not needed.

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